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Search	Most Recent Queries	Time	Result
#6	Search (gp140) and (infection) AND (hiv) AND ("CCR5" [TIAB] OR "CCR-5" [TIAB] OR "CCR5" [TIAB] OR "C-C chemokine receptor type 5" [TIAB] OR "CCCKR5" [TIAB] OR "CCCKR-5" [TIAB] OR "CCCKR 5" [TIAB] OR "C-C CKR-5" [TIAB] OR "CC-CKR-5" [TIAB] OR "CCR-5" [TIAB] OR "CD195" [TIAB] OR "CD-195" [TIAB] OR "CD 195" [TIAB] OR "CD195 antigen" [TIAB] OR "CHEMR13" [TIAB] OR "CHEMR-13" [TIAB] OR "CHEMR 13" [TIAB] OR "CKR5" [TIAB] OR "CKR-5" [TIAB] OR "CKR 5" [TIAB] OR "CKR-5" [TIAB] OR "CMKBR5" [TIAB] OR "CMKBR-5" [TIAB] OR "CMKBR 5" [TIAB] OR "HIV-1 fusion coreceptor" [TIAB]) [TIAB]	13:22:23	<u>9</u>
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Symbol	Name	Synonyms	Organism
CCR5	chemokine (C-C motif) receptor 5	C-C chemokine receptor type 5, CCCR5, CC-CKR-5, C-C CKR-5, CCR-5, CD195, CD195 antigen, CHEMR13, CKR5, CKR-5, CMKBR5, HIV-1 fusion coreceptor	Homo sapiens


UniProt P51681, O14708, O14699
 IntAct P51681
 PDB Structure 1NE0, 1OPN
 OMIM 601373
 NCBI Gene 1234
 NCBI RefSeq NP_000570
 NCBI RefSeq NM_000579
 NCBI UniGene 1234
 NCBI Accession CAA62796, BC038398


Homologues of CCR5 ... **new**


Interaction information for this gene  ...


Enhanced PubMed/Google query ... **new**


The enhanced macrophage tropism correlated with reduced sensitivity to inhibition by Q4120, a CD4-specific antibody, but not with sensitivity to the **CCR5**  inhibitor, TAK779.


To modulate migration, human macrophages were incubated in the presence of aminooxypentane-regulated on activation, normal, T-cell expressed, and secreted (AOP-RANTES), a potent antagonist of **CCR5** .


Prototype HIV-1 isolates from the CNS are macrophage (M)-tropic, non-syncytia-inducing (NSI), and use **CCR5**  for entry (R5 strains), but whether syncytia-inducing (SI) CXCR4-using X4 strains might play a role in macrophage/microglia infection and neuronal injury is unknown.


The small-molecule **CCR5**  antagonist SCH-C (SCH 351125) was tested for its ability to inhibit HIV-1 replication in peripheral blood mononuclear cells (PBMCs), cord blood mononuclear cells, immature dendritic cells (DCs), and macrophages.


G protein-dependent **CCR5**  signaling is not required for efficient infection of primary T lymphocytes and macrophages by R5 human immunodeficiency virus type 1 isolates.


Targeting **CCR5**  with siRNAs: using recombinant SV40-derived vectors to protect macrophages and microglia from R5-tropic HIV.

To understand host mechanisms that affect human immunodeficiency virus type 1 (HIV-1) pathogenesis by modulating expression of coreceptors, cytokine regulation of CC chemokine receptor 5 (**CCR5** ) and CD4 expression on monocytes, monocyte-derived macrophages (MDMs), and microglia was investigated.

Hemofiltrate CC chemokine 1[9-74] causes effective internalization of **CCR5**  and is a potent inhibitor of R5-tropic human immunodeficiency virus type 1 strains in primary T cells and macrophages.

In the present study we demonstrate that HCC-1[9-74] interacts with the second external loop of **CCR5**  and inhibits replication of CCR5-tropic HIV-1 strains in both primary T cells and monocyte-derived macrophages.

CCR5  surface expression was absent on T lymphocytes and macrophages.

Human cytomegalovirus infection reduces surface **CCR5**  expression in human microglial cells, astrocytes and monocyte-derived macrophages.

Such binding was dependent on cell surface glycosaminoglycans (GAGs) since it was reduced when macrophages or HeLa cells expressing or not **CCR5**  were first treated with GAG-specific enzymes.

CONCLUSION: Opiates enhance HIV R5 strain infection of macrophages through the downregulation

of beta-chemokine production and upregulation of CCR5 receptor [?] ☆ expression and may have an important role in HIV immunopathogenesis.

Coreceptor use in transfected cells generally predicted use in primary macrophages, although for some Envs macrophages may be a more sensitive indicator of CCR5 ☆ use than transfected cell lines.

Macrophages infiltrating the tissue in chronic pancreatitis express the chemokine receptor CCR5 [?] ☆.

Expression of CCR5 ☆ is increased in human monocyte-derived macrophages and alveolar macrophages in the course of in vivo and in vitro Mycobacterium tuberculosis infection.

Because infection of macrophages and microglial cells by NSI HIV-1 is considered to be instrumental for the development of AIDS dementia complex (ADC), we studied whether the CCR5 ☆ Delta32 heterozygous genotype correlated with a reduced frequency of ADC.

CCR5 ☆- and CXCR4-positive macrophages and microglia were detected in inflammatory lesions in the brain of children with severe HIV.

Prostaglandin E2 induces resistance to human immunodeficiency virus-1 infection in monocyte-derived macrophages: downregulation of CCR5 ☆ expression by cyclic adenosine monophosphate.

Alanine substitutions of polar and nonpolar residues in the amino-terminal domain of CCR5 ☆ differently impair entry of macrophage- and dualtropic isolates of human immunodeficiency virus type 1.

Recent discovery of co-receptor, chemokine receptor (CCR5 ☆) which is expressed in macrophages, may give a clue to understand the mechanism of HIV encephalopathy more precisely.

FTY-induced lymphopenia preferentially affects CD62L+ and CCR5 ☆- T-lymphocyte subpopulations.

CONCLUSIONS: Our data suggest an involvement of CCR5 ☆ in T-cell accumulation in the inflamed central nervous system.

Novel reporter T-cell line highly susceptible to both CCR5 ☆- and CXCR4-using human immunodeficiency virus type 1 and its application to drug susceptibility tests.

To establish a simple and rapid assay system for the monitoring of R5 HIV-1 replication and drug susceptibility, we have established a novel reporter T-cell line, MOCHA (which represents MOLT-4 cells stably expressing CCR5 ☆ and carrying the HIV-1 long terminal repeat-driven secretory alkaline phosphatase).

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Symbol	Name	Synonym/ DB-reference	Organism
		Life cycles of successful g	
CCR5	chemokine (C-C motif) receptor 5		Homo sapiens